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The primary visual cortex, and feedback to it, are not necessary for conscious vision

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A compelling single case report of visual awareness (visual qualia) without primary visual cortex would be sufficient to refute the hypothesis that the primary visual cortex and the back-projections to it are necessary for conscious visual experience. In a previous study, we emphasized the presence of crude visual awareness in Patient G.Y., with a lesion of the primary visual cortex, who is aware of, and able to discriminate, fast-moving visual stimuli presented to his blind field. The visual nature of Patient G.Y.'s blind field experience has since been questioned and it has been suggested that the special circumstances of repeated testing over decades may have altered Patient G.Y.'s visual pathways. We therefore sought new evidence of visual awareness without primary visual cortex in patients for whom such considerations do not apply. Three patients with hemianopic field defects (Patient G.N. and Patient F.B. with MRI confirmed primary visual cortex lesions, Patient C.G. with an inferred lesion) underwent detailed psychophysical testing in their blind fields. Visual stimuli were presented at different velocities and contrasts in two- and four-direction discrimination experiments and the direction of motion and awareness reported using a forced-choice paradigm. Detailed verbal reports were also obtained of the nature of the blind field experience with comparison of the drawings of the stimulus presented in the blind and intact fields, where possible. All three patients reported visual awareness in their blind fields. Visual awareness was significantly more likely when a moving stimulus was present compared to no stimulus catch trials ($P < 0.01$ for each subject). Psychophysical performance in Patient F.B. and Patient G.N. was consistent with the Riddoch syndrome, with higher levels of visual awareness for moving compared to static stimuli ($P < 0.001$) and intact direction discrimination ($P < 0.0001$ for two- and four-direction experiments). Although the blind field experience of all three subjects was degraded, it was clearly visual in nature. We conclude that the primary visual cortex or back-projections to it are not necessary for visual awareness.

Introduction

In the work reported here, we confine ourselves to addressing a single question: is the primary visual cortex (V1), and the return input to it from other cortical sources, necessary for conscious

vision? Since the publication of our previous papers, in which we demonstrated that a subject with damage to V1 can still have conscious awareness of (fast) motion presented in his blind field (Barbur et al., 1993; ffytche et al., 1996; Zeki and ffytche, 1998), several publications have affirmed that a feedback to V1,

and hence a healthy V1, are necessary for conscious vision (Lamme, 2001; Pascual-Leone and Walsh, 2001; Tong, 2003; Silvanto et al., 2005a, b; Silvanto, 2008). Indeed, there are theories positing the notion that only when the feedback system to V1 is operating can what has been processed visually acquire a conscious correlate (Lamme and Roelfsema, 2000; Lamme, 2001). There are many experiments of various levels of sophistication that address the question of whether a return input to V1 is necessary for visual consciousness. We believe that none of these are compelling since the only convincing demonstration of the truth or falsity of this proposition is to enquire whether a person blinded by a lesion in V1 can still be aware of a visual stimulus presented to his blind field. Experiments that artificially deactivate V1 or area V5 of the visual cortex reversibly (Silvanto et al., 2005a, b) or study the artificial production of phosphenes following stimulation of V1 or V5 (Cowey and Walsh, 2000; Pascual-Leone and Walsh, 2001) do not provide compelling and direct evidence in this regard. Similarly, experiments demonstrating a lack of awareness of visual stimulation in patients blinded by a lesion to V1 do not provide a compelling argument for or against the involvement of V1 or back-projections to it, since the correct task to elicit awareness among the infinite number of possible tasks may not have been used. But a positive answer, of the presence of visual awareness in a patient with loss of V1, assuming that all other criteria regarding lesion location are met, would be conclusive evidence to show that V1 and a return projection to it are not necessary for visual awareness, which is not the same thing as saying that V1 is not important or does not enrich visual experience and awareness.

Providing confirmation of this supposition is not difficult. All that is required is a human subject with a V1 lesion large enough to produce a hemianopia and evidence of visual awareness, with the proviso that there is no spared tissue within the damaged V1 that could plausibly account for the awareness. If such a subject, when presented with visual stimuli in the blind field, reports perceiving a stimulus that meets acceptable criteria of visual awareness, then it is obvious that neither V1 nor the return input to it are necessary. In this work, we are not concerned with the level of sophistication of the awareness, our only concern being with its visual nature and its presence or absence in response to a visual stimulus presented to the blind field. Our criterion for visual awareness does not differ much from the common sense view that might be given by a lay person: namely if they verbalize that they can see it and draw it, and if the drawing resembles the stimulus that they perceive and draw when presented in their intact field, then we consider the patient to have been visually aware during the presentation of the stimulus. We have previously published evidence of visual awareness in the absence of V1 (Barbur et al., 1993; ffytche et al., 1996; Zeki and ffytche, 1998), a finding that has been confirmed by others (Morland et al., 1999; Stoerig and Barth, 2001). Yet the evidence in all these studies has been based on the single patient, Patient G.Y., and while the presence of awareness in Patient G.Y.'s blind field has been conceded (Weiskrantz et al., 1995), its 'visual' nature has been questioned on the grounds that extensive training may have led him to develop an awareness of visual stimulation through a circuitous route (Cowey, 2004) or that his awareness is metamodal and not visual (Pascual-Leone and Walsh, 2001) or lacks visual

qualia (Persaud and Lau, 2008) (see the 'Discussion' section). We felt it important, therefore, to seek other patients to confirm or refute the role of V1 in visual awareness. Our conclusion from three new cases in the work reported below is that, contrary to previous suppositions and confirming our view of the visual nature of awareness in Patient G.Y., V1 is not necessary for a crude but conscious visual awareness of visual occurrences in a blind field, and hence neither are the return projections to it.

Materials and methods

Subjects

Subjects were self-referred to S.Z. ($n = 2$) or through their clinical teams ($n = 1$). Each gave informed consent for visual testing and the studies were approved by the UCL/UCH joint ethics committee (ref 98/N82).

Patient G.N. is a 26-year-old Italian male who suffered a head injury while on holiday in Dublin at the age of 17 years, following an assault that resulted in an extradural haemorrhage with herniation of the temporal lobes, compromised posterior cerebral artery circulation and a right occipital stroke. He remained in a coma for several weeks after the injury. On recovery, he had a dense left hemianopia without macular sparing and post-traumatic parkinsonism related to midbrain/thalamic infarcts. He has difficulty in walking and initiating mouth and lip movements, with marked dysarthria. He communicates with his carer using sign language and keyboards/telephone keypads. His cognitive abilities are relatively intact and he is able to understand English, Italian and Spanish, spending much of his time corresponding on the internet. Patient G.N. was tested 9 years after his occipital stroke.

Figure 1A shows Patient G.N.'s perimetric examination with a dense left homonymous hemianopia 7 years after the injury. His right hemispheric lesion (Fig. 1B and C) selectively damaged the medial and polar occipital surface involving the upper and lower banks of the calcarine fissure and lingual gyrus. Anteriorly, the lesion extends to the parahippocampal gyrus and the lateral occipital cortex is spared. The location of the lesion with respect to the calcarine fissure indicates loss of V1 and ventral areas V2/V3 of the visual cortex.

Patient F.B. is a 64-year-old French male with hypertension and hypercholesterolaemia, with a right hemianopia following a left occipital lobe stroke. At the time of testing, 6 months after the stroke, he had a small strip of spared vision in his right superior quadrant at the vertical meridian that was particularly apparent on testing his right eye (Fig. 2A). The stroke affected the medial and ventral left occipital surface indicating loss of V1 and ventral areas V2/V3 of the visual cortex (Fig. 2B) and the lateral occipital cortex was spared.

Patient C.G. is a 57-year-old London caterer with a dense left homonymous hemianopia without macular sparing following a right hemispheric stroke (Fig. 3). His only other symptom was a persistent intense tinnitus ('like Concord') and he had difficulty reading. Patient C.G. was tested 1 year after his occipital stroke and magnetic resonance images of his lesion are not available.

Visual testing

Patients G.N. and F.B. were tested in their homes in Turin and Paris, respectively, while Patient C.G. was tested at University College London. Patients G.N. and F.B. were tested with a Gaussian circular disc presented at different velocities and contrasts in their blind fields
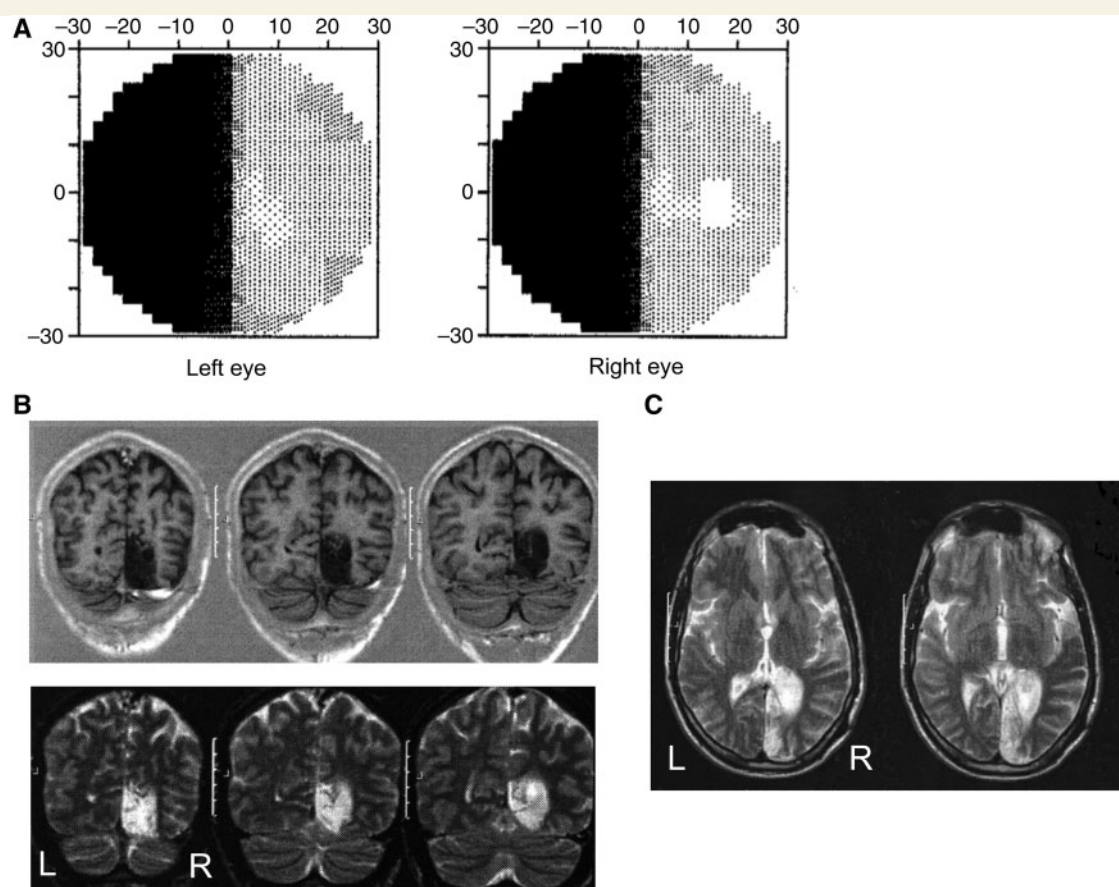
Figure 1 Patient G.N. (**A**) The visual field in Patient G.N.'s left and right eye. The black region shows the hemianopic left visual field with involvement of the macular region. Scale = 30° horizontal and vertical eccentricity. (**B**) Coronal MRI slices showing the extent of the lesion in Patient G.N.'s right occipital lobe. The lesion involves the medial and polar surface of the right hemisphere, and includes the upper and lower banks of the calcarine fissure and lingual gyrus but spares the lateral occipital surface. (**C**) Axial MRI slices of the lesion showing its anterior extension to the parahippocampal gyrus.

(Table 1). Stimuli were presented on a laptop with a liquid crystal display, calibrated for luminance and timing, using an in-house software stimulus presentation programme. Both subjects were asked to maintain fixation on a cross on the screen. Because Patients G.N. and F.B. were tested in their homes abroad, we did not have the eye-monitoring equipment that we would have otherwise used. Instead, in a subset of experiments one of us monitored fixation by facing the subject behind the laptop and observing their eyes throughout each trial. No eye movements were detected during these monitored experiments, confirming that the results described below are not attributable to eye movements or failures of fixation. A mask of maximum intensity was presented in the intact field extending into the blind field by 1–3.5° to prevent detection of scattered light from the blind to the intact hemifield. Each experiment consisted of a block of 20 trials (total number of trials Patient G.N. = 260; Patient F.B. = 180). A moving stimulus was presented in 16 trials and a static one in two; there were two 'no stimulus' catch trials. The order of trials and directions was randomized within each block. Separate blocks of trials presented moving stimuli in two or four directions (up/down, left/right or up/down and left/right). Each trial was initiated by the experimenter after establishing that the subject was ready. Stimuli were presented after a random delay (1–3 s). To minimize onset and offset transients, the stimulus faded-in and -out over 200–500 ms (fade time was held

constant within an experiment); the stationary stimulus had the same fade-in and -out times. For each block, velocity, contrast, location and trajectory were held constant. The trajectory was also held constant for fast and slow-motion experiments so that an identical region of the visual field was stimulated in the two conditions. For Patient F.B. the target in vertical trajectory experiments started (downward motion) or ended (upward motion) close to the lower margin of the region of spared visual field in the right superior quadrant; however, the fade-in and -out of the stimulus meant that the target did not enter the spared region. After completion of each trial, the subject was required to give the direction of motion and answer two questions related to awareness: (i) whether he had seen the stimulus; and (ii) a confidence rating of seeing (or not seeing) the stimulus (Table 2). Based on our previous study (Zeki and ffytche, 1998), the confidence rating was simplified to a two-point scale, certain or unsure, with the former testifying to the presence of awareness unambiguously. The awareness and confidence questions in combination gave four possible outcomes: (i) seen/certain; (ii) seen/unsure; (iii) not seen/unsure; and (iv) not seen/certain. Two definitions of awareness were used in the analysis, a more lenient definition capturing any uncertainty as to the presence or absence of a stimulus [outcomes (i–iii) = 'aware' trials; outcome (iv) = 'unaware' trials] and a more stringent definition only accepting trials reported 'seen' as aware [outcomes (i and ii) = 'aware' trials;
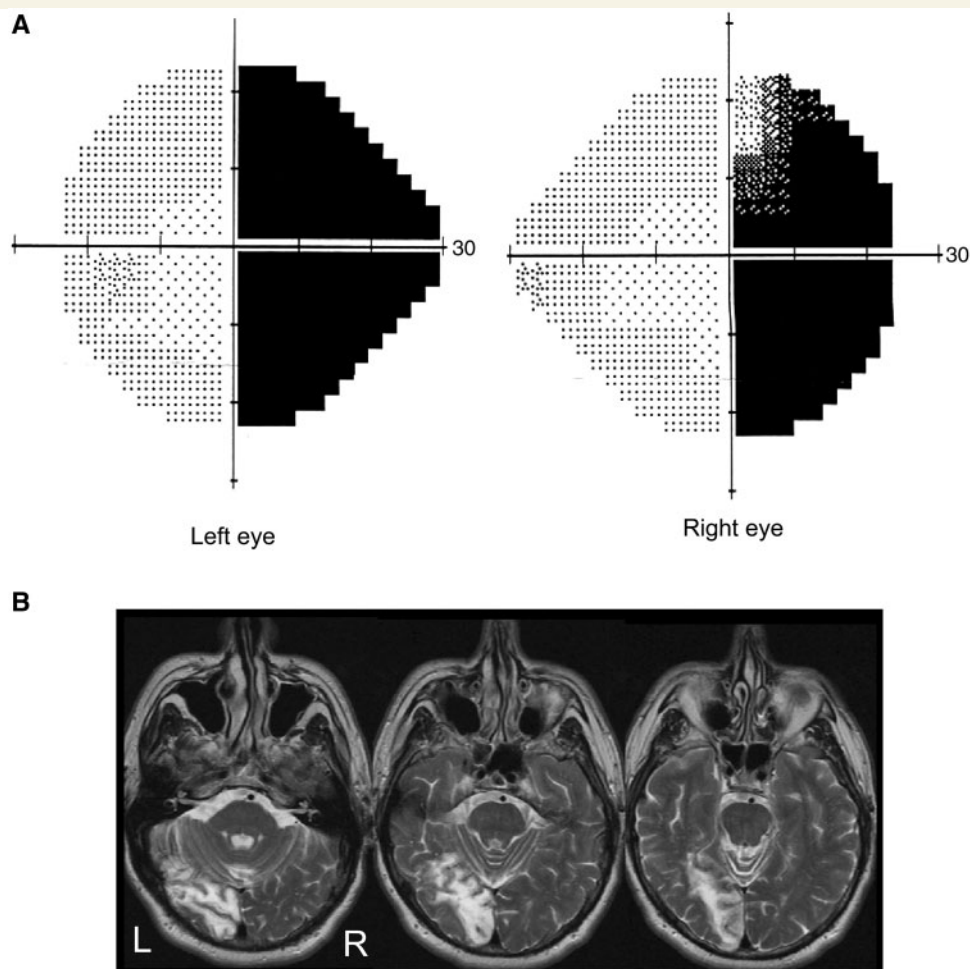
Figure 2 Patient F.B. (**A**) The visual field in Patient F.B.'s left and right eye. The black region shows the hemianopic right visual field with sparing at the vertical meridian in the right superior quadrant. Scale = 30° horizontal and vertical eccentricity. (**B**) Axial MRI slices showing the extent of Patient F.B.'s lesion. It involves the medial and polar surface of the left occipital lobe but spares its lateral surface.
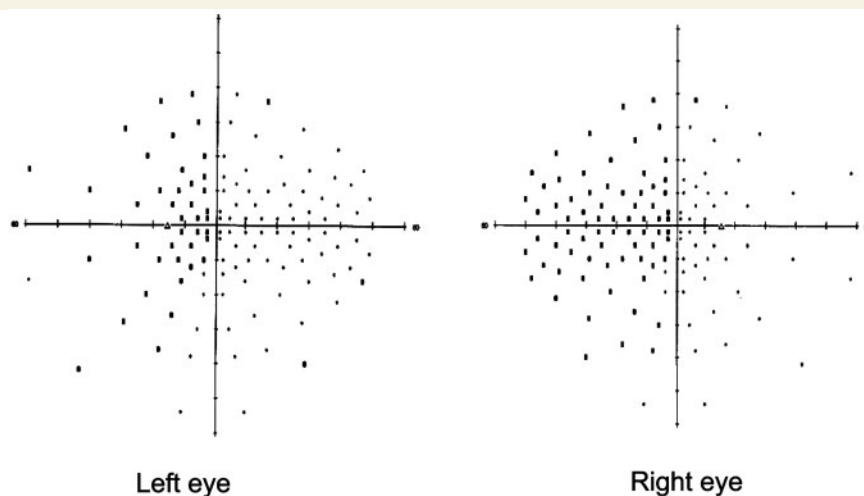

Figure 3 The visual fields in Patient C.G.'s left and right eye. Darker black bars indicate areas of field loss. Patient C.G. has a left hemianopia involving the macular region with some sparing of the left lower quadrant at the vertical meridian.

Table 1 Summary of experimental stimuli

Subject	#	Location[a]	Target diameter	Directions	Trajectory	Velocity (°/s)	Contrast (%)	Trials
Patient G.N.	1	5°	5°	Four directions	26°	6°	66	20
	2	14°	5°	Four directions	26°	6°	89	20
	3	14°	5°	Four directions	26°	6°	89	20
	5	14°	5°	Four directions	26°	18°	89	20
	6	14°	5°	Four directions	26°	18°	89	20
	7	14°	5°	Four directions	26°	18°	89	20
	9	17°	5°	Four directions	16°	19°	89	20
	10	17°	5°	Up/down	16°	20°	89	20
	11	10°	5°	Up/down	19°	20°	89	20
	12	10°	5°	Up/down	19°	2°	89	20
	13	10°	5°	Up/down	19°	25°	89	20
	14	10°	5°	Up/down	19°	25°	56	20
	15	10°	5°	Up/down	19°	2°	56	20
Patient F.B.	1	7°	5°	Up/down	19°	29°	32	20
	2	7°	5°	Up/down	19°	29°	74	20
	3	3°	3°	Up/down	19°	29°	80	20
	5	3°	3°	Up/down	14°	2°	32	20
	6	3°	3°	Up/down	14°	27°	32	20
	7	3°	3°	Up/down	14°	28°	20	20
	8	3°	3°	Up/down	14°	2°	20	20
	9	7.6° V = 11°	3°	Left/right	8°	25°	20	20
	10	7.6° V = 11°	3°	Left/right	8°	26°	81	20
Patient C.G.	5	7°	8°	Up/down	5°	8°	73	10
	10	7°	8°	Up/down	5°	8°	73	80
	15	9°	8°	Up/down	5°	8°	62	200
	16	9°	8°	Up/down	5°	8°	62	100

a Midpoint of trajectory lies on the horizontal meridian at eccentricity given. Two blocks of trials for Patient F.B. were presented in the upper quadrant (V = vertical eccentricity).

refers to the order of the experiments. Incomplete experiments are not reported. Patient C.G. also took part in shape and colour discrimination experiments not reported here. Shaded rows indicate experiments used to examine the relationship between discrimination and awareness for fast and slow motion (Fig. 5).

Table 2 Response questions and forced choices

Question	Response choice
Did you see any motion?	Yes/no
How confident are you?	Certain/unsure
What was the direction?	Two- or four-direction forced choice

outcomes (iii and iv) = 'unaware' trials]. We did not attempt to map systematically different regions of the visual field; however, small variations in stimulus location occurred between some of the experiments (Table 1) occasioned, for example, by the change from four- to two-direction experiments or following initial training.

Patient C.G. underwent a similar testing procedure to Patients G.N. and F.B., with the difference that stimulus contrast was deliberately reduced to bring his performance close to the threshold of motion detection. Like Patients G.N. and F.B., he was presented with a circular stimulus in his blind hemifield that could be moving (up/down), be stationary or be a catch trial (the proportion of stationary trials varied between 20% and 38% in different experiments, catch trials varied between 25% and 50%). After each trial, Patient C.G. was asked to report the direction of motion and answer awareness and confidence questions (total number of trials = 390). Since

Patient C.G. always reported the stimulus to be a stationary flash, whether it was moving or not, and therefore denied seeing motion (see 'Results' section) his awareness responses were restricted to 'not seen' and 'unsure'. Instead of a two-point confidence scale he was asked to give a percentage rating of confidence for each unsure response (from 0% = no confidence to 100% = fully confident). In the experiments he only used confidence ratings of between 5% and 50% and these trials were deemed 'aware' in the analysis to capture even the slightest evidence of awareness. Trials reported 'not seen' were deemed 'unaware'.

Statistical analysis

The number of moving stimuli reported as 'aware' was compared with the number of catch or stationary stimuli reported as 'aware' using χ^2-tests. Scores for all experiments in each subject (Table 1) were pooled for this analysis. The discrimination of motion direction in the blind field was examined using binomial theorem to calculate the likelihood of the percentage correct score occurring by chance. In Patients G.N. and F.B., a subset of two-direction experiments in which velocity and contrast were varied systematically (see shaded rows in Table 1) were compared with our previously published model of psychophysical performance in the Riddoch syndrome (Zeki and ffytche, 1998).

Phenomenological analysis

While familiarizing themselves with the task and its requirements, subjects were interviewed after each trial regarding the nature of their experience. Once the task was fully understood, they reported their experiences using the forced-choice awareness and confidence responses. At the end of the experiments, they were re-interviewed about the nature of the experience in their blind field and asked to compare it to that in their intact field. Where possible they were asked to draw the experiences in their blind and intact fields.

Results

All three subjects reported visual experiences (i.e. visual qualia) in their blind hemifields when presented with moving stimuli. Below we describe the phenomenology of these experiences and the psychophysical performance associated with them.

Awareness

All three subjects showed a significantly higher percentage of aware trials in their hemianopic fields for moving stimuli compared with catch trials (Table 3), only rarely reporting awareness when no stimulus was presented. For Patients G.N. and F.B., aware trials were almost always reported 'seen' (response categories seen/certain and seen/unsure) with only 0.3% of trials reported by Patient G.N. and 8.3% of trials reported by Patient F.B. as 'not seen' (response category not seen/unsure pooled across fast motion, slow motion, blank and catch trials). The percentage of motion trials with awareness in the three subjects varied from 57% to 74% reflecting the fact that low contrast, slow-motion trials were included in the analysis. If only fast motion, high-contrast trials are included in the analysis, almost all motion trials were aware (Patient G.N. = 97% aware for motion trials in Experiments 5–7, 11 and 13; Patient F.B. = 93% aware for motion trials in Experiments 3 and 6; Patient C.G. did not perform experiments with high-contrast stimuli). Patients G.N. and F.B. also had a significantly higher percentage of aware trials for moving compared with static stimuli, consistent with the performance expected of patients with the Riddoch syndrome (Zeki and ffytche, 1998). In contrast, there was no difference in the percentage of awareness for moving or stationary stimuli in Patient C.G., suggesting a different type of deficit (see below). For Patients G.N. and F.B., the stringency of the definition of awareness made little difference to the results (Table 4) as both rarely reported they were uncertain that they had not seen a trial (not seen/uncertan responses), in other words when reporting that they had not seen the stimulus they were almost always certain that they had not seen it (response = not seen/certain).

Phenomenology

All three subjects were able to describe visual experiences in their blind field. Patients G.N. and F.B. were able to draw their experiences (Fig. 4). Patient G.N.'s ability to communicate verbally is limited by his dysarthria; however, he was able to report (both verbally and by typing on a keypad) that there was little

Table 3 Percentage awareness for each stimulus category

	Motion aware	Catch aware	Static aware	Total trials
Patient G.N.	72.5%	11.5%**	30.8%**	259
Patient F.B.	57.3%	16.7%*	11.1%**	179
Patient C.G.	74.8%	0%**	80.7%	390

$**P < 0.001$, $*P < 0.01$, χ^2-test versus motion aware.

Table 4 Percentage awareness (stringent definition) for each stimulus category

	Motion aware	Catch aware	Static aware	Total trials
Patient G.N.	72.5%	11.5%**	26.9%**	259
Patient F.B.	50.3%	0%**	0%**	179

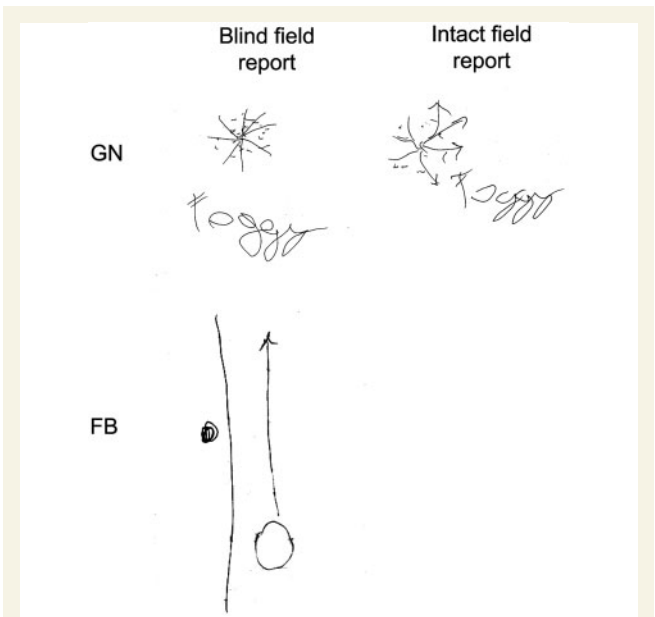$**P < 0.001$, χ^2-test versus motion aware.

Figure 4 Phenomenology. Patient G.N. and Patient F.B.'s drawings of the experiences in their blind hemifields. Patient G.N. was also asked to draw his experience of the same stimulus when presented in his intact field for comparison. Both subjects are able to depict their experiences visually as well as provide a verbal report of their visual nature.

distinction between the visual experience in his intact and blind hemifields, describing the Gaussian disc as 'foggy' in both hemifields. Patient F.B. was able to describe seeing a shape that moved in his blind field and to draw it. The drawing relates to the whole trajectory of the stimulus, that is to say it includes the hemianopic region extending from above the horizontal meridian to the lower quadrant. Patient C.G. reported seeing a flash whenever he was aware of a stimulus. He defined a flash as 'Just a change in the light, nothing else . . . It's like you are sitting there (Patient C.G.

puts one hand on the table like a screen and places his other hand behind it) and there is something behind and something flashes. When you are sat there you can see the flash but you can't see what is behind, what caused it . . .'.

Discrimination of motion direction

For high contrast, fast-motion trials, Patient G.N. (Experiments 5–7, 11 and 13) and Patient F.B. (Experiments 3 and 6) were able to discriminate motion direction with close to 100% accuracy (Patient G.N.: two-direction experiments 96% correct, $P < 0.0001$; four-direction experiments 77% correct, $P < 0.0001$; Patient F.B.: two-direction experiments 90% correct, $P < 0.0001$). Patient C.G. was not able to discriminate motion direction (two-direction experiments 47% correct, $P = 0.25$), since he could only report the presence of a 'flash' whenever a stimulus appeared. We examined the relationship of Patient G.N. and Patient F.B.'s performance to our previous psychophysical model of the Riddoch syndrome, using a subset of experiments that assessed up/down discrimination at different contrasts and velocities in the same portion of the visual field (see shaded cells in Table 1 for experiments used). Figure 5 shows that both subjects (Patient F.B. = squares, Patient G.N. = circles) follow the same function whereby the percent correct score is explained by the percent aware trials + the proportion of unaware trials

Figure 5 Discrimination and awareness for fast and slow motion. Percent correct responses are plotted against percent aware responses for up/down experiments in Patients G.N. (circles) and F.B. (squares) (shaded rows in Table 1). Slow-motion trials are shown in red, fast-motion trials in blue. The black line indicates the predicted performance of patients with the Riddoch syndrome with the upper and lower limits of chance at $P < 0.05$ given by the dotted lines (Zeki and ffytche, 1998). The red square labelled 'high' is the high contrast, slow-motion experiment for Patient F.B.

correct by chance (black and dotted lines) (Zeki and ffytche, 1998). Psychophysical performance tended to lie to the left of the hypothetical function (black line), a shift we termed 'gnosanopsia' in our previous article, reflecting the fact that discrimination scores were worse than might be expected from awareness scores. There was no evidence of an ability to discriminate without awareness [termed 'agnosopsia' in our previous article and renamed blindsight type 1 by Weiskrantz (1998a, b)]. Just as in our previous study, the percent correct and percent aware scores for slow motion (red) were lower than those for fast motion (blue), with the exception of the high contrast, slow-motion experiment in one block of trials for Patient F.B. (red square labelled high) where the percent correct and percent aware scores were better than expected. We attribute this anomalous finding to his region of spared visual field (see below).

Discussion

Our concern in this work centres on the single question of whether V1 or feedback to it is 'necessary' for visual consciousness. This would not be so if patients blinded by a lesion in V1 were able to (consciously) perceive a stimulus presented to their hemianopic fields. We had shown this to be true in 1993 for the most extensively studied subject, Patient G.Y. (Barbur et al., 1993), and our demonstration was confirmed by Weiskrantz et al. (1995), with further studies by Morland et al. (1999) and Stoerig and Barth (2001), attesting to the visual nature of his blind field experience. However, the nature of Patient G.Y.'s awareness has subsequently been questioned, prompting us to look for new evidence elsewhere.

Location and extent of damage to the primary visual cortex

We first address the question of the location and extent of the lesions in the three cases studied. In this report, we have inferred the involvement of V1 by relying on (i) perimetric studies that have charted the extent of the blind fields; and (ii) structural images obtained by magnetic resonance, which in Patient G.N. was taken several years after the initial damage and in Patient F.B., after 6 months. Both show that the lesions were in V1, although they extended beyond it. One possible objection might be derived from the arguments used by Fendrich et al. (1992), namely that, in spite of the hemianopia, preserved islands of tissue within V1, not readily detectable with structural scans and conventional perimetry, may have mediated the conscious vision that we and others report. Indeed, a recent imaging study suggests that a map of the contralateral visual field may be preserved in lesioned V1 (Radoeva et al., 2008), although the lesion in this case is only apparent in the immediate aftermath of a stroke and appears resolved at the time of the mapping experiments. We discount this explanation in the case of Patient G.N., for the following reason. He (like Patient F.B.) scored more poorly for the slow than the fast motion task. If the ability to discriminate a stimulus, and visual awareness of it, are based on an island of preserved V1, we would have expected the opposite pattern of results with better performance and awareness for the
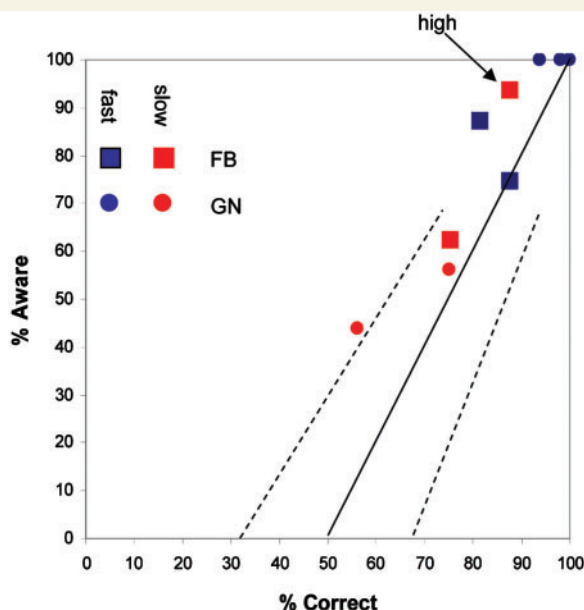
slow-motion task than the fast motion task as the slow-moving stimulus is of longer duration and hence affords greater opportunity for detection by a spared island of vision. The fact that fast moving stimuli in Patients G.N. and F.B. (as well as Patient GY in our previous study) were associated with higher levels of awareness and discrimination provides compelling evidence against the role of spared V1 islands. Furthermore, our EEG/magnetoencephalography experiments suggest that signals from slow-moving stimuli ($<6°/s$) are relayed to V1 before reaching area V5 of the visual cortex (ffytche *et al.*, 1995), whereas signals from fast moving stimuli reach area V5 before reaching V1, so that an island of spared V1 would be expected to preferentially detect slow-moving stimuli. For the same reason, we believe that the 'island' argument does not apply to Patient F.B. either, even though there were variations in the degree of impairment across his visual field (see below). The only finding in Patient F.B. consistent with the island account is that high contrast, slow motion was in one block of trials associated with better awareness and discrimination scores than high contrast, fast motion, which could reflect the increased likelihood of the longer lasting slow-motion stimulus straying into spared areas of vision. Perhaps equally important in arguing against the island account is the phenomenal experience of Patients G.N. and F.B. (and Patient G.Y.), which is not that of discontinuous, insular and piece-meal vision, as might be predicted if their vision were related to an island of spared cortex, but of a continuous trajectory of motion. The only subject in whom the phenomenology is consistent with an island was Patient C.G., who reported all stimulation, whether stationary or moving, as resulting in a stationary flash. It is possible that Patient C.G. did not perceive continuous motion—unlike Patient G.N., Patient F.B. and Patient G.Y.—because his visual awareness was limited to an island of spared vision, although we would not wish to speculate further on this.

Although we did not systematically map the hemianopic visual field we note, like Fendrich *et al.* (1992), that the degree of blindness was not uniform in one of our subjects (Patient F.B.), with subregions where the number of aware trials decreased and discrimination performance fell to chance levels. For example, at 7° (Experiment 1) his performance was at chance ($P=0.1$) and his awareness at 0% aware while at 3° (Experiment 6) it was 81% correct ($P < 0.005$) and 87% aware. We have no ready explanation for this, but suggest that it may be due to unequal damage to subcortical pathways projecting directly to area V5 of the visual cortex from different parts of the visual field, since area V5 receives visual inputs that bypass V1 (Cragg, 1969; Yukie and Iwai, 1981; Standage and Benevento, 1983; Sincich *et al.*, 2004; Schmid *et al.*, 2010).

Crude visual awareness in the blind field

We then looked at the question of whether our subjects were visually aware in their blind fields. We had no reason to doubt their reports of awareness on statistical grounds. Awareness was significantly more likely to be reported when a stimulus was present compared with when it was not, with few false reports of awareness when no stimulus was present. Patients G.N. and F.B. were also significantly more likely to report awareness when the stimulus was moving compared with when static, but also (with the exception of one, anomalous, block of trials for Patient F.B.) when the motion was fast compared with slow, defining characteristics of the Riddoch syndrome. Equally important is the additional demonstration that the subjects could prepare drawings of what they had perceived in their blind field, which compare favourably with the drawings of the same stimuli when presented to their intact fields. We did not ask our subjects to draw their experiences after every trial; however, we assume that every time a subject reported seeing the stimulus using the forced-choice responses, they had the type of experience depicted in their drawings and described in their verbal report. These descriptions left us in no doubt that the experiences they had were visual in nature and amounted to what might be called 'visual qualia' and considerable doubts in accounting for them in other ways. This is not to say that the blind field experiences are identical to those in the intact field or to understate the poverty of the blind visual experiences. Indeed, Riddoch originally described his subjects as experiencing the moving stimuli in their blind fields as 'shadowy', stating that they are sure that 'they can attribute neither form nor colour' to them (Riddoch, 1917). This is also similar to the description given by Gordon Holmes, who was otherwise generally hostile to the description given by Riddoch, that his Subject 11 was '….generally conscious only of the movement of the white test object, and saw it only "as through a mist", and as a "dirty grey colour"…..' (Holmes, 1918). In fact, when we first asked Patient G.Y. about his phenomenal experience, he described it in terms of shadows and thus in a way remarkably similar to the descriptions given by Riddoch. It is only more recently that Patient G.Y. has used the term 'feeling' to qualify his visual experience (Zeki and ffytche, 1998), a term that none of our present patients used. Our point is simply that certain types of visual stimuli presented to the blind field are undeniably associated with a crude visual awareness. It should be noted that on a small percentage of trials, Patients G.N. and F.B. reported that they did not see the stimulus but were not confident about this (not seen/unsure responses). This type of response occurred both for catch and stationary trials as well as for motion trials, making it unlikely that it reflects non-visual awareness of the stimulus or a metamodal alerting response as has been proposed for Patient G.Y. (Pascual-Leone and Walsh, 2001). We interpret these responses in our subjects as a consequence of task expectations.

Our results in relation to 'blindsight'

We next address our results in the context of blindsight, a term originally defined as meaning an intact capacity to discriminate with a very high accuracy visual stimuli presented to the blind field but without an acknowledged visual awareness (Sanders *et al.*, 1974). As the review entitled 'The blindsight saga' by Cowey (2010) shows, 'blindsight' remains highly controversial. Why should a syndrome described over 35 years ago remain so controversial today? The answer is not far to seek. On the one hand, as Cowey's review shows, there has been no conclusive demonstration of the phenomenon and every description of it

has met with objections. On the other hand, each challenge has entailed a changing of the goal posts. The acknowledgment (Weiskrantz et al., 1995) that subjects blinded by lesions in V1 can be aware of the presence and direction of motion of fast moving (>6°/s), high-contrast visual stimuli (Barbur et al., 1993; Zeki and ffytche, 1998) led to a re-definition of blindsight into two categories Blindsight 1 and 2, (Weiskrantz, 1998a, b), the latter corresponding to conditions in which the subject is both aware of visual stimuli and able to discriminate them. More recently, further variants termed 'action blindsight' and 'attention blindsight' have emerged (Danckert and Rossetti, 2005). The demonstration that Patient G.Y., the most studied patient in this regard, can match the stimulus presented in his blind field with that presented in his intact field (Morland et al., 1999; Stoerig and Barth, 2001), thus supporting the view that the patient has a crude visual awareness of what is presented to his blind hemifield, is countered by the suggestion that the extensive training and testing of patients can lead to the adoption of bizarre and highly sophisticated strategies (Cowey, 2004) while the demonstration that he is aware of the fast moving stimuli that he discriminates is explained by the fact that these are not moving stimuli at all but a displacement of stimuli from one position to another (non-Fourier feature tracking—see Azzopardi and Cowey, 2001). All this is capped by the suggestion that the reported consciousness of patients with prolonged testing, like Patient G.Y., is not of a visual nature, though elicited by a visual stimulus, but of autonomic (pupil) and skeletal (eye muscle) responses, reflected in variations in pupillary size or eye movements, in other words that patients become aware of their eye movements or the dilatation of their pupils, but not of the visual stimulus itself. In fact, a recent study suggests the opposite is more typical of patients with V1 lesions, as patients trained intensively to detect motion in their blind fields evolved from 'sensing' the stimulus in the early stages of training to 'actively seeing a proportion of the dots' as training progressed (Huxlin et al., 2009). Finally, for good measure, the doubtful assumption that V1 is indispensible for visual awareness calls into question theoretical accounts of consciousness based on networks and 'top-down' processing (Cowey, 2010). In our view, a stable phenomenon should not need to rely on ever changing criteria and definitions. That the criteria keep changing explains, to a significant extent, its present highly controversial status (Cowey, 2010). We believe that arguments based on awareness of pupil size or eye movements, instead of the presence of the visual stimulus, are far too sophisticated and unnecessary, especially when subjects can match the characteristics of the visual stimulus presented to their blind fields with that presented to their intact fields. The visual awareness reported by the three patients presented here is also not attributable to the type of intensive training reported by Huxlin et al. (2009), since they had not been tested prior to this study. Furthermore, all our subjects acquired their lesions in later life, thus arguing against substantial rewiring of their visual pathways as has been suggested for subjects with spared blind field visual capacities following surgical removal of a hemisphere as treatment for lesions present at birth or in early infancy (Leh et al., 2006) or subjects with full visual fields despite congenital absence of occipital cortex (Werth, 2006). Recent diffusion tensor tractography studies of Patient G.Y. (virtual

dissections of white matter based on probabilistic measures) are consistent with a substantial rewiring of his visual pathways (Bridge et al., 2008). Such evidence, if correct, raises doubts as to whether Patient G.Y. can be used to infer the normal functions of V1 and also calls into question the validity of all previous conclusions with respect to the neurobiology of visual awareness and the phenomenon of 'blindsight' derived from this patient.

In addition to awareness of the stimuli, two of the subjects were able to discriminate their direction in a manner entirely consistent with that previously described for Patient G.Y. (Zeki and ffytche, 1998). Patient G.N. and Patient F.B.'s percent correct and percent aware scores followed the same function as found in Patient G.Y. with higher rates of aware and correct responses for fast motion compared with slow motion and for high contrast compared with low-contrast stimuli. We account for these observations by supposing that a visual input to area V5 of the visual cortex, through either the superior colliculus–pulvinar pathway, or through the lateral geniculate nucleus [both of which have been demonstrated to project to V5 without relaying in V1 (Yukie and Iwai, 1981; Standage and Benevento, 1983), with recent evidence that the lateral geniculate nucleus pathway is particularly important in this regard (Schmid et al., 2010)], is sufficient to elicit a crude but conscious visual experience of a moving stimulus as well as some of its characteristics. The pathway seems to convey inputs related to fast moving and visually salient (high contrast) stimuli (ffytche et al., 1995, 1996; Morand et al., 2000). Moreover, our imaging experiments (Barbur et al., 1993; Zeki and ffytche, 1998) show that activity in area V5 correlates with the experience of fast moving stimuli in Patient G.Y. blinded by a lesion to V1. The recent experiment of Schmid et al. (2010) in which they lesioned V1 in the monkey and showed that signals from the lateral geniculate nucleus can bypass V1 to be relayed directly to area V5, confirms the conclusions reached in our previous human work (ffytche et al., 1995, 1996; Zeki and ffytche, 1998).

Crude conscious vision in the absence of primary visual cortex and back-projections to it

Our findings from two of three subjects blinded by lesions in V1 thus lead to the conclusion that conscious visual awareness is possible without V1 or back-projections to it. The findings from the third subject (Patient C.G.) may also be similarly interpreted but are also open to other interpretations, namely the presence of spared islands in V1 (Fendrich et al., 1992). We repeat that this is not tantamount to saying that vision in subjects with such lesions is normal, or that back-projections to V1 or forward projections from it are not important for normal vision, nor do we pretend that area V5 of the visual cortex can fulfil its task without other enabling pathways, for example from the brainstem (Zeki and ffytche, 1998). Our sole contention is that a back-projection to V1 is not mandatory for visual consciousness.

Some recent transcranial magnetic stimulation papers have suggested otherwise (Cowey and Walsh, 2000; Pascual-Leone and Walsh, 2001; Silvanto et al., 2005a, b; Silvanto, 2008), but we do not consider their evidence compelling. Studies employing

transcranial magnetic stimulation inactivation do not have the same status, in our view, as our current studies because they constitute an artificial means of stimulation in otherwise healthy subjects, without knowing the precise extent of cortical and white matter stimulation. The details in these papers make them even less compelling. In the article of Pascual-Leone and Walsh (2001), transcranial magnetic stimulation was effective in only 8 of 26 subjects. We trace this inefficacy to the fact that the stimulus was delivered to a grid of locations centred 4 cm from the midline, although details of the stimulation point for each individual are not given. The centre point of the grid used by Pascual-Leone and Walsh (2001) is posterior to the estimated position of area V5 in our transcranial magnetic stimulation study (Beckers and Zeki, 1995) and this, together with the ineffectiveness of stimulation in the majority of subjects (18 of the 26 tested), suggests they may have been stimulating another cortical location, possibly area V3 of the visual cortex. The key observation in the study by Silvanto *et al.* (2005*a, b*) of two time points in which the deactivation of V1 led to a deficit in sensitivity to a change in the stimulus (subjects were not asked to discriminate the direction of motion) was made by joining two different experiments, involving different subjects. That study also deactivated the cortex 88 ms after stimulus onset, well after the arrival of signals in either area V5 or V1 (ffytche *et al.*, 1995). Moreover, it used a stimulus speed (2°/s) that would be expected to be influenced by V1 inactivation, since as we have previously shown (ffytche *et al.*, 1995) only fast moving stimuli access V5 without going through V1, whereas slow-moving stimuli ($<$6°/s) are channelled to V5 through V1.

V1, which receives a massive input from the retina through the lateral geniculate nucleus, has extensive reciprocal connections with many of the visual areas surrounding it. Various functions have been attributed to these return connections, including binding, both within the separately processed visual attributes (e.g. figure-ground segregation) and between them (see Lamme and Roelfsema, 2000; Roelfsema, 2006 for reviews), and binding itself has been considered to be crucial for consciousness (Crick and Koch, 2003). That these feedback connections can bind the responses of cells processing different attributes seems plausible, given that the feedforward projections from V1 to areas of the visual cortex such as V4 and V5 are highly segregated, whereas the return input from them to V1 distribute not only to the territory of cells in V1 that project to them but also to the territory of cells that project elsewhere, thus allowing for cells in a specialized area to influence cells in V1 projecting to other specialized areas (Zeki and Shipp, 1988; Shipp and Zeki, 1989). If binding leads to the emergence of a conscious correlate, it seems reasonable to suppose that feedback projections to V1 are essential for binding, and thus consciousness. However, although these are important arguments for a critical role for feedback connections to V1 in generating a conscious visual experience, they do not demonstrate that such feedback connections are essential.

In fact, the arguments against the supposition that feedback connections are essential for generating a conscious experience are also largely suggestive. They revolve around the fact that there are direct inputs to areas of the visual cortex such as V4 and V5 bypassing V1, making it plausible to suppose that they can

deliver signals to the specialized visual area whose activity can then generate a conscious correlate for these attributes without the mandatory involvement of V1. They also revolve around the fact that, at least over very brief time frames, binding may not be necessary for generating a conscious correlate, since separate attributes appear to be processed and perceived at different times by the brain, leading to the notion that there are many separate visual consciousnesses (Moutoussis and Zeki, 1997). These arguments, though suggestive, are also not compelling in showing that a return feedback to V1 is not necessary for conscious vision.

Conclusion

Whatever the merits of these arguments for or against a critical role for feedback to V1 in generating a conscious experience of the visual world, they are not determining ones. The only direct evidence in support of one argument or the other is the demonstration that a subject with a lesion in V1 can have a conscious if crude visual experience. We have demonstrated that this is so for the patient who has been most extensively studied in the past, Patient G.Y. (Barbur *et al.*, 1993; Zeki and ffytche, 1998) and have critically examined antecedent evidence to show that most previous results are in accord with our view in spite of different interpretations given (Zeki and ffytche, 1998). As it happens, there is now considerable evidence to support our conclusion (Morland *et al.*, 1999; Kleiser *et al.*, 2001; Stoerig and Barth, 2001; Overgaard *et al.*, 2008), which is also supported by earlier papers, even those purportedly demonstrating the absence of visual experiences (see Table 4 in Zeki and ffytche, 1998).

Lamme and colleagues (2000) have strongly emphasized that, while a fast feed-forward system can process a visual stimulus, a return feedback to V1 is necessary for the subject to be consciously aware of the stimulus (Lamme and Roelfsema, 2000). We do not dispute any of this, nor do we question the importance of either a healthy V1 or a healthy return input to it for normal visual experience. Feedback systems in the cortex have been thought of as being important for a number of characteristics, including error-coding, modifying activity in an 'earlier' area or subjecting an earlier area to influences that render it more capable of completing its task of processing. Our sole contention is that a feedback specifically to V1 is not necessary for a conscious visual experience. We do not include other visual cortical areas in our conclusion, and do not extend it to question the importance of feedback in generating conscious experiences at large in the cortex.

That a crude but conscious experience of vision is possible without V1, and hence without a feedback input to V1, naturally leaves us with the puzzle of how an impoverished input to area V5 of the visual cortex can result in a conscious visual percept, however crude, and how this conscious percept is generated.

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